Decision Augmentation Theory: Toward a Model of Anomalous Mental Phenomena

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Abstract

Decision Augmentation Theory (*DAT*) holds that humans integrate information obtained by anomalous cognition into the usual decision process. The result is that, to a statistical degree, such decisions are biased toward volitional outcomes. We introduce *DAT* and define the domain for which the model is applicable. In anomalous mental phenomena research, *DAT* is applicable to the understanding of effects that are within a few sigma of chance. We contrast the experimental consequences of *DAT* with those of models that treat anomalous perturbation as a causal force. We derive mathematical expressions for *DAT* and causal models for two distributions, normal and binomial. *DAT* is testable both retrospectively and prospectively, and we provide statistical power curves to assist in the experimental design of such tests. We show that the experimental consequences of *DAT* are different from those of causal models except for one degenerate case.

Introduction

We do not have positive definitions of the effects that generally fall under the heading of anomalous mental phenomena (*AMP*).* In the crassest of terms, *AMP* is what happens when nothing else should, at least as nature is currently understood. In the domain of information acquisition, or anomalous cognition (*AC*), it is relatively straightforward to design an experimental protocol (Honorton et al., 1990, Hyman and Honorton, 1986) to assure that no known sensory leakage of information can occur. In the domain of causation, or anomalous perturbation (*AP*), however, it is very difficult, if not impossible (May, Humphrey, and Hubbard, 1980 and Hubbard, Bentley, Pasturel, and Issacs, 1987); thus, making the interpretation of results equally difficult.

We can divide *AP* into two categories based on the magnitude of the putative effect. Macro-*AP* include phenomena that generally do not require sophisticated statistical analysis to tease out weak effects from the data. Examples include inelastic deformations in strain gauge experiments, the obvious bending of metal samples, and a host of possible "field phenomena" such as telekinesis, poltergeist, teleportation, and materialization. Conversely, micro-*AP* covers experimental data from noisy diodes, radioactive decay and other random sources. These data show small differences from chance expectation and require statistical analysis.

One of the consequences of the negative definitions of *AMP* is that experimenters must assure that the observables are not due to "known" effects. Traditionally, two techniques have been employed to guard against such interactions:

(1) Complete physical isolation of the *AP*-target system.
(2) Counterbalanced control and effort periods.

Isolating physical systems from potential "environmental" effects is difficult, even for engineering specialists. It becomes increasingly problematical the more sensitive the Macro-*AP* device. For example Hubbard, Bentley, Pasturel, and Issacs (1987) monitored a large number of sensors of environmental variables that could mimic *AP* effects in an extremely isolated piezoelectric strain gauge. Among these were three-axis accelerometers, calibrated microphones, and electromagnetic and nuclear radiation monitors. In addition, the sensors were mounted in a government-approved enclosure to assure no leakage (in or out) of electromagnetic radiation above a given frequency, and the enclosure itself was levitated on an air suspension table. Finally, the entire setup was locked in a controlled access room which was monitored by motion detectors. The system was so sensitive, for example, that it was possible to identify the source of a perturbation of the strain gauge that was due to innocent, gentle knocking on the door of the closed room. The financial and engineering resources to isolate such systems rapidly become prohibitive.

The second method, which is commonly in use, is to isolate the target system within the constraints of the available resources, and then construct protocols that include control and effort periods. Thus, we trade complete isolation for a statistical analysis of the difference between control and effort periods. The assumption implicit in this approach is that environmental influences of the device will be random

* The Cognitive Sciences Laboratory has adopted the term *anomalous mental phenomena* instead of the more widely known *psi*. Likewise, we use the terms anomalous cognition and anomalous perturbation for *ESP* and *PK*, respectively. We have done so because we believe that these terms are more naturally descriptive of the observables and are neutral with regard to mechanisms. These new terms will be used throughout this paper.

and uniformly distributed in both the control and effort conditions, while *AP* will tend to occur in the effort periods. Our arguments in favor of an anomaly, then, are based on statistical inference and we must consider, in detail, the consequences of such analyses, one of which implies a generalized model for *AMP*.

Background

As the evidence for *AMP* becomes more widely accepted (Bem and Honorton, 1994, Utts, 1991, Radin and Nelson, 1989) it is imperative to determine the underlying mechanisms of the phenomena. Clearly, we are not the first to begin thinking of potential models. In the process of amassing incontrovertible evidence of an anomaly, many theoretical approaches have been examined; in this section we outline a few of them. It is beyond the scope of this paper, however, to provide an exhaustive review of the theoretical models of *AMP*; a good reference to an up-to-date and detailed presentation is Stokes (1987).

Brief Review of Models

Two fundamentally different types of models have been developed: those that attempt to order and structure the raw observations in *AMP* experiments (i.e., phenomenological), and those that attempt to explain *AMP* in terms of modifications to existing physical theories (i.e., fundamental). In the history of the physical sciences, phenomenological models, such as the Snell's law of refraction or Ampere's law for the magnetic field due to a current, have nearly always preceded fundamental models of the phenomena, such as quantum electrodynamics and Maxwell's theory. In producing useful models of *AMP* it may well be advantageous to start with phenomenological models, of which *DAT* is an example.

Psychologists have contributed interesting phenomenological approaches. Stanford (1974a and 1974b) proposed *PSI*-mediated Instrumental Response (PMIR) as a descriptive model. PMIR states that an organism uses *AMP* to optimize its environment. For example, in one of Stanford's classic experiments (Stanford, Zenhausern, Taylor, and Dwyer 1975) subjects were offered a covert opportunity to stop a boring task prematurely if they exhibited unconscious *AP* by perturbing a hidden random number generator. Overall, the experiment was significant in the unconscious tasks; it was as if the participants were unconsciously scanning the extended environment for any way to provide a more optimal situation than participating in a boring psychological task!

As an example of a fundamental model, Walker (1984) proposed a literal interpretation of quantum mechanics in that since superposition of eigenstates holds, even for macrosystems, *AMP* might be due to macroscopic examples of quantum phenomena. These concepts spawned a class of theories, the so-called observation theories, that were based either upon quantum formalism conceptually or directly (Stokes, 1987). Jahn and Dunne (1986) have offered a "quantum metaphor" which illustrates many parallels between *AMP* and known quantum effects. Unfortunately, these models either have free parameters with unknown values, or are merely hand waving metaphors and therefore have not led to testable predictions. Some of these models propose questionable extensions to existing theories. For example, even though Walker's interpretation of quantum mechanical formalism might suggest wave-like properties of macrosystems, the physics data to date not only show no indication of such phenomena at room temperature but provide considerable evidence to suggest that macrosystems lose their quantum

coherence above 0.5 Kelvins (Washburn and Webb, 1986) and no longer exhibit quantum wave-like behavior.

This is not to say that a comprehensive model of *AMP* will not eventually require quantum mechanics as part of its explanation, but it is currently premature to consider such models as more than interesting speculation. The burden of proof is on the theorist to show why systems, which are normally considered classical (e.g., a human brain), are, indeed, quantum mechanical. That is, what are the experimental consequences of a quantum mechanical system over a classical one?

Our Decision Augmentation Theory is phenomenological and is a logical and formal extension of Stanford's elegant PMIR model. In the same manner as early models of the behavior of gases, acoustics, or optics, it tries to subsume a large range of experimental measurements into a coherent lawful scheme. Hopefully this process will lead the way to the uncovering of deeper mechanisms. In fact *DAT* leads to the idea that there may be only one underlying mechanism of all *AMP* effects, namely a transfer of information between events separated by negative time intervals.

Historical Evolution of Decision Augmentation

May, Humphrey, and Hubbard (1980) conducted a careful random number generator (RNG) experiment. What makes this experiment unique is the extreme engineering and methodological care that was taken in order to isolate any potentially known physical interactions with the source of randomness. It is beyond the scope of this paper to describe this experiment completely; however, those specific details which led to the idea of Decision Augmentation are important for the sake of historical completeness.

May, Humphrey, and Hubbard were satisfied in that RNG study, that they had observed a genuine statistical anomaly. In addition, because of an accurate mathematical model of the random device and the engineering details of the experiment, they were equally satisfied that the deviations were not due to any known physical interactions. They concluded, in their report, that some form of *AMP*-mediated data selection had occurred. They named it then *Psychoenergetic Data Selection.*

Following a suggestion by Dr. David R. Saunders of MARS Measurement and Associates, we noticed in 1986 that the effect size in binary RNG studies varied on the average as the square root of the number of bits in the sequence. This observation led to the development of the *Intuitive Data Sorting* model that appeared to describe the RNG data to that date (May, Radin, Hubbard, Humphrey, and Utts, 1985).

The remainder of this paper describes the next step in the evolution process. We now call the model *Decision Augmentation Theory* (*DAT*).

Decision Augmentation Theory-A General Description

Since the case for *AC*-mediated information transfer is now well established, it would be exceptional if we did *not* integrate this form of information gathering into the decision process. For example, we routinely use real-time data gathering and historical information to assist in the decision process. Perhaps, what is called intuition may play a important role. Why, then, should we not include *AC* information?

DAT holds that *AC* information is included along with the usual inputs that result in a final human decision that favours a "desired" outcome. In statistical parlance, *DAT* says that a slight, systematic bias is introduced into the decision process by *AC*.

This philosophical concept has the advantage of being quite general. We know of no experiment that is devoid of at least one human decision; thus, *DAT* might be the underlying basis for *AMP*. To illustrate the point, we describe how the "cosmos" determines the outcome of a well-designed, hypothetical experiment. To determine the sequencing of an RNG experiment, suppose that the entry point into a table of random numbers will be chosen by the square root of the barometric pressure as stated in the weather report that will be published seven days hence in the *New York Times*. Since humans are notoriously bad at predicting or controlling the weather, this entry point might seem independent of a human decision; but why did we "chose" seven days in advance? Why not six or eight? Why the *New York Times* and not the *London Times*? *DAT* would suggest that the selection of seven days, the *New York Times*, the barometric pressure, and square root function were optimal choices, either individually or collectively, and that other decisions would not lead to as significant an outcome.

Other non-technical decisions may also be biased by *AC* in accordance with *DAT*. When should we schedule a Ganzfeld session; who should be the experimenter in a series; how should we determine a specific order in a tri-polar protocol?

It is important to understand the domain in which a model is applicable. For example, Newton's laws are sufficient to describe the dynamics of mechanical objects in the domain where the velocities are very much smaller than the speed of light, and where the quantum wavelength of the object is very small compared to the physical extent of the object. If these conditions are violated, then different models must be invoked (e.g., relativity and quantum mechanics, respectively).

The domain in which *DAT* is applicable is when experimental outcomes are in a statistical regime (i.e., a few standard deviations from chance). In other words, does the measured effect occur under the null hypothesis? This is not a sharp-edged requirement and *DAT* becomes less apropos the more a single measurement deviates from mean-chance-expectation (MCE). We would not invoke *DAT*, for example, as an explanation of levitation if one found the authors hovering near the ceiling!

All this may be interesting philosophy, but *DAT* can be formulated mathematically and subjected to rigorous examination.

Development of a Formal Model

While *DAT* may have implications for *AMP* in general, we develop the model in the framework of understanding experimental results. In particular, we consider *AP* vs *AC* in the form of *DAT* in those experiments whose outcomes are in the few-sigma, statistical regime.

We define four possible mechanisms for the results in such experiments:

(1) **Mean Chance Expectation.** The results are at chance. That is, the deviation of the dependent variable meets accepted criteria for MCE. In statistical parlance, we have measurements from an *unperturbed* parent distribution with *unbiased* sampling.

(2) **Anomalous Perturbation.** Nature is modified by some anomalous interaction. That is, we expect a causal interaction of a "force" type. In statistical parlance, we have measurements from a *perturbed* parent distribution with *unbiased* sampling.

(3) **Decision Augmentation.** Nature is unchanged but the measurements are biased. That is, *AC* information has "distorted" the sampling. In statistical parlance, we have measurements from an *unperturbed* parent distribution with *biased* sampling.

(4) **Combination.** Nature is modified and the measurements are biased. That is, both *AP* and *AC* are present. In statistical parlance, we have conducted *biased* sampling from a *perturbed* parent distribution.

General Considerations

Since the formal discussion of *DAT* is statistical, we will describe the overall context for the development of the model from that perspective. Consider a random variable, X, that can take on continuous values (e.g., the normal distribution) or discrete values (e.g., the binomial distribution). Examples of X might be the hit rate in an RNG experiment, the swimming velocity of cells, or the mutation rate of bacteria. Let Y be the average computed over n values of X, where n is the number of items that are *collectively* subjected to an *AMP* influence as the result of a single decision—one trial. Often this may be equivalent to a single effort period, but it also may include repeated efforts. The key point is that, regardless of the effort style, the average value of the dependent variable is computed over the n values resulting from one decision point. In the examples above, n is the sequence length of a single run in an RNG experiment, the number of swimming cells measured during the trial, or the number of bacteria-containing test tubes present during the trial.

Assumptions for DAT

We assume that the parent distribution of a physical system remains *unperturbed*; however, the measurements of the physical system are systematically biased by some *AC*-mediated informational process.

Since the deviations seen in experiments in the statistical regime tend to be small in magnitude, it is safe to assume that the measurement biases might also be small; therefore, we assume small shifts of the mean and variance of the sampling distribution. Figure 1 shows the distributions for biased and unbiased measurements.

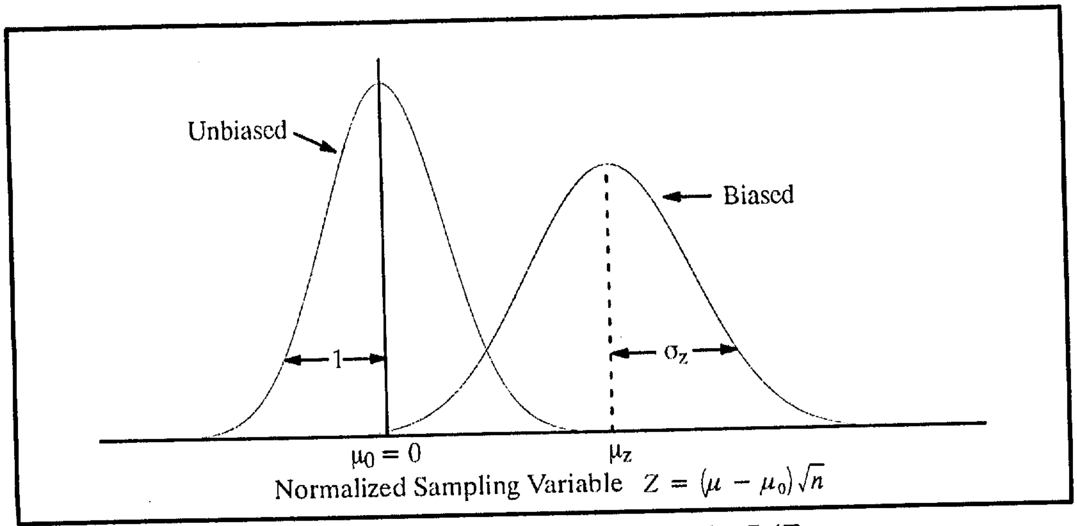

Figure 1. Sampling Distribution Under *DAT*.

The biased sampling distribution shown in Figure 1 is assumed to be normally distributed as:

$$Z \sim N(\mu_z, \sigma_z^2),$$

where the notation means that Z is distributed as a normal distribution with a mean of μ_z and a standard deviation of σ_z.

Assumptions for an AP Model

For comparison sake, we develop a model for AP interactions. With a few exceptions reported in the poltergeist literature, AP appears to be a relatively "small" effect in laboratory experiments. That is, we do not readily observe anomalous and obvious mental interactions with the environment. Thus, we begin with the assumption that a putative AP force would give rise to a perturbational interaction. What we mean is that given an ensemble of entities (e.g., binary bits, cells), a force acts, on the average, equally on each member of the ensemble. We call this type of interaction perturbational AP (PAP).

Figure 2 shows a schematic representation of probability density functions for a parent distribution under the PAP assumption and an unperturbed parent distribution. In the PAP model, the perturbation induces a change in the mean of the parent distribution but does not effects its variance. We parameterize the mean shift in terms of a multiplier of the initial standard deviation. Thus, we define an AP-effect size as:

$$\varepsilon_{AP} = \frac{(\mu_1 - \mu_0)}{\sigma_0},$$

where μ_1 and μ_0 are the means of the perturbed and unperturbed distributions, respectively, and where σ_0 is the standard deviation of the unperturbed distribution.

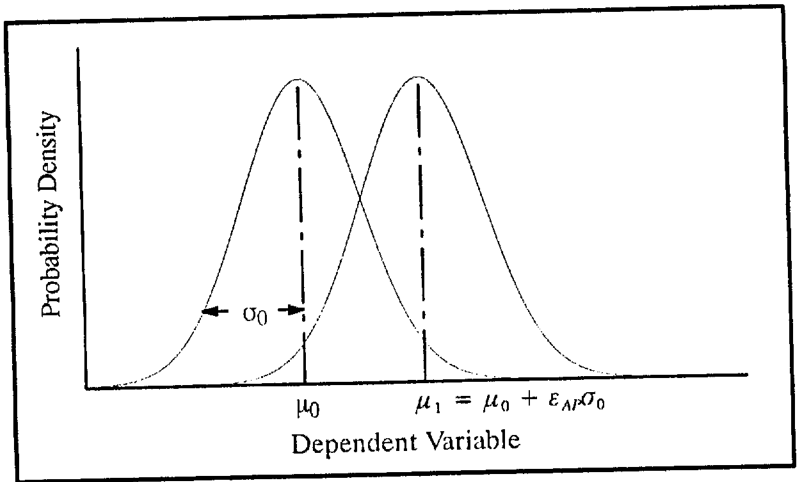

Figure 2. Parent Distribution for Perturbational AP.

For the moment, we consider ε_{AP} as a parameter which, in principle, could be a function of a variety of variables (e.g., psychological, physical, environmental, methodological). As we develop DAT for specific distributions and experiments, we will discuss this functionality of ε_{AP}.

Calculation of E(Z^2)

We compute the expected value and variance of Z^2 under MCE, PAP, and DAT for the normal and binomial distributions. The details of the calculations can be found in the Appendix; however, we summarize the results in this section. Table 1 shows the results assuming that the parent distribution is normal.

Table 1.

Normal Parent Distribution

Quantity	Mechanism		
	MCE	PAP	DAT
$E(Z^2)$	1	$1 + \varepsilon_{AP}^2 n$	$\mu_z^2 + \sigma_z^2$
$Var(Z^2)$	2	$2(1 + 2\varepsilon_{AP}^2 n)$	$2(\sigma_z^4 + 2\mu_z^2\sigma_z^2)$

Table 2 shows the results assuming that the parent distribution is binomial. In this calculation, p_0 is the binomial event probability and $\sigma_0 = \sqrt{p_0(1-p_0)}$.

Table 2.

Binomial Parent Distribution

Quantity	Mechanism		
	MCE	PAP	DAT
$E(Z^2)$	1	$1 + \varepsilon_{AP}^2(n-1) + \frac{\varepsilon_{AP}}{\sigma_0}(1 - 2p_0)$	$\mu_z^2 + \sigma_z^2$
$Var(Z^2)$	$2 + \frac{1}{n\sigma_0^2}(1 - 6\sigma_0^2)$	$2(1 + 2\varepsilon_{AP}^2 n)^*$	$2(\sigma_z^4 + 2\mu_z^2\sigma_z^2)$

* The variance shown assumes $p_0 = 0.5$ and $n \gg 1$. See the Appendix for other cases.

We wish to emphasize at this point that in the development of the mathematical model, the parameter ε_{AP} for *PAP*, and the parameters μ_z, and σ_z in *DAT* may all possibly depend upon n; however, for the moment, we *assume* that they are all n-independent. We shall discuss the consequences of this assumption below.

Figure 3 displays these theoretical calculations for the three mechanisms graphically.

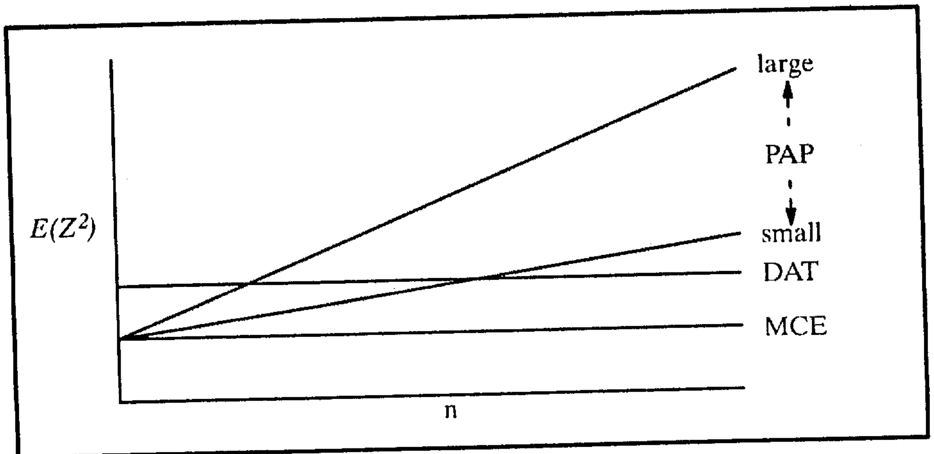

Figure 3. Predictions of *MCE, PAP,* and *DAT.*

Within the constraints mentioned above, this formulation predicts grossly different outcomes for these models and, therefore, is ultimately capable of separating them, even for very small perturbations.

Retrospective Tests

It is possible to apply *DAT* retrospectively to any body of data that meet certain constraints. It is critical to keep in mind the meaning of n—the number of measures of the dependent variable over which to compute an average during a single trial following a single decision. In terms of their predictions for experimental results, the crucial distinction between *DAT* and the *PAP* model is the dependence of the results upon n; therefore, experiments which are used to test these theories must be those in which experiment participants are blind to n. In a follow-on to this theory-definition paper, we will retrospectively apply *DAT* to as many data sets as possible, and examine the consequences of any violations of these criteria.

Aside from these considerations, the application of *DAT* is straight forward. Having identified the unit of analysis and n, simply create a scatter diagram of points (Z^2, n) and compute a least square fit to a straight line. Tables 1 and 2 show that for the *PAP* model, the square of the *AP*-effect size is the slope of the resulting fit. A student's t-test may be used to test the hypothesis that the *AP*-effect size is zero, and thus test for the validity of the *PAP* model. If the slope is zero, these same tables show that the intercept may be interpreted as an *AC* strength parameter for *DAT*. The follow-on paper will describe these techniques in detail.

Prospective Tests

A prospective test of *DAT* will not only test the *AMP* hypothesis against mean chance expectation, but will also test for a *PAP* contribution. In such tests, n should certainly be a double-blind parameter and take on at least two values. If you wanted to check the prediction of a linear functional relationship between n and the $E(Z^2)$ that is suggested by *PAP* model, the more values of n the better. It is not possible to separate the *PAP* model from *DAT* at a single value of n.

In any prospective test, it is helpful to know the number of runs, N, that are necessary to determine with 95% confidence, which of the two models best fits the data. Figure 4 displays the problem graphically.

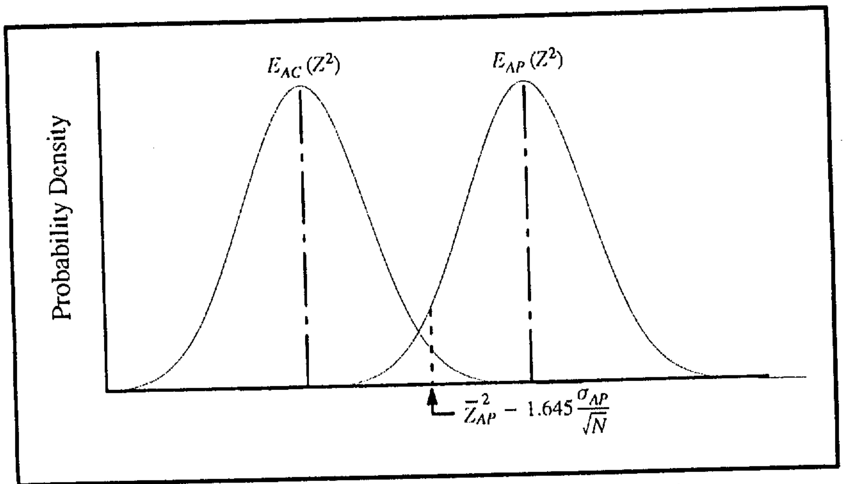

Figure 4. Model Predictions for the Power Calculation.

Under *PAP*, 95% of the values of $\overline{Z}^2$ will be greater than the point indicated in Figure 4. Even if the measured value of $\overline{Z}^2$ is at this point, we would like the lower limit of the 95% confidence interval for this value to be greater than the predicted value under the *DAT* model. Or:

$$\overline{Z}^2_{AP} - 1.645\frac{\sigma_{AP}}{\sqrt{N}} - 1.960\frac{\sigma_{AP}}{\sqrt{N}} \geq E_{AC}(Z^2).$$

Solving for N in the equality, we find:

$$N = \left[\frac{3.605\ \sigma_{AP}}{E_{AP}(Z^2) - E_{AC}(Z^2)}\right]^2. \qquad (1)$$

Since $\sigma_{AP} \geq \sigma_{AC}$, this value of N will always be the larger estimate than that derived from beginning with *DAT* and calculating the confidence intervals in the other direction.

Suppose, from an earlier experiment, one can estimate a single-trial effect size for a specific value of n, say n_1. To determine whether the *PAP* model or *DAT* is the proper description of the mechanism, we must conduct another study at an additional value of n, say n_2. We use Equation 1 to compute how many runs we must conduct at n_2 to assure a separation of mechanism with 95% confidence, and we use the variances shown in Tables 1 and 2 to compute σ_{AP}. Figure 5 shows the number of runs for an RNG-like experiment as a function of effect size for three values of n_2.

We chose $n_1 = 100$ bits because it is typical of the numbers found in the RNG database and the values of n_2 shown are within easy reach of today's computer-based RNG devices. For example, assuming $\sigma_z = 1.0$ and assuming an effect size of 0.004, one we derived from a publication of PEAR data (Jahn, 1982), then at $n_1 = 100, \mu_z = 0.004 \times \sqrt{100} = 0.04$ and $E_{AC}(Z^2) = 1.0016$. Suppose $n_2 = 10^4$. Then $E_{AP}(Z^2) = 1.160$ and $\sigma_{AP} = 1.625$. Using Equation 1, we find $N = 1368$ runs, which can be approximately obtained from Figure 5. That is in this example, 1368 runs are needed to resolve the *PAP* model from *DAT* at $n_2 = 10^4$ at the 95% confidence level. Since these runs are easily obtained in most RNG experiments, an ideal prospective test of *DAT*, which is based on these calculations, would be to conduct 1500 runs randomly counterbalanced between $n = 10^2$ and $n = 10^4$ bits/trial. *If* the effect size at $n = 10^2$ is near 0.004, than we would resolve the *AP* vs *AC* question with 95% confidence.

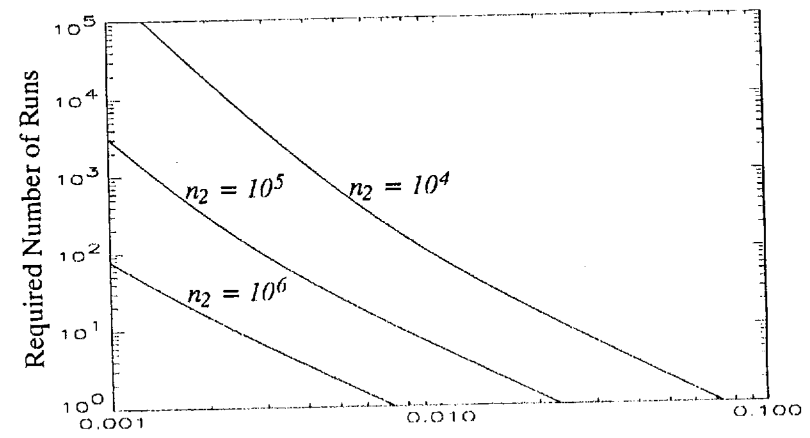

Figure 5. Runs Required for RNG Effect Sizes

Figure 6 shows similar relationships for effect sizes that are more typical of biological *AP* as reported in the Former Soviet Union (May and Vilenskaya, 1994).

Similarly, for biological oriented *AP* experiments, we chose $n_1 = 2$ because use two simultaneous *AP* targets is easily accomplished. If we assume an effect size of 0.3 and $\sigma_z = 1.0$, at $n_2 = 10$ we compute $E_{AC}(Z^2) = 1.180$, $E_{AP}(Z^2) = 1.900$, $\sigma_{AP} = 2.366$ and $N = 140$, which can be approximately obtained from Figure 6.

We have included $n_2 = 100$ in Figure 6, because this is within reach in cellular experiments although it is probably not practical for most biological *AP* experiments.

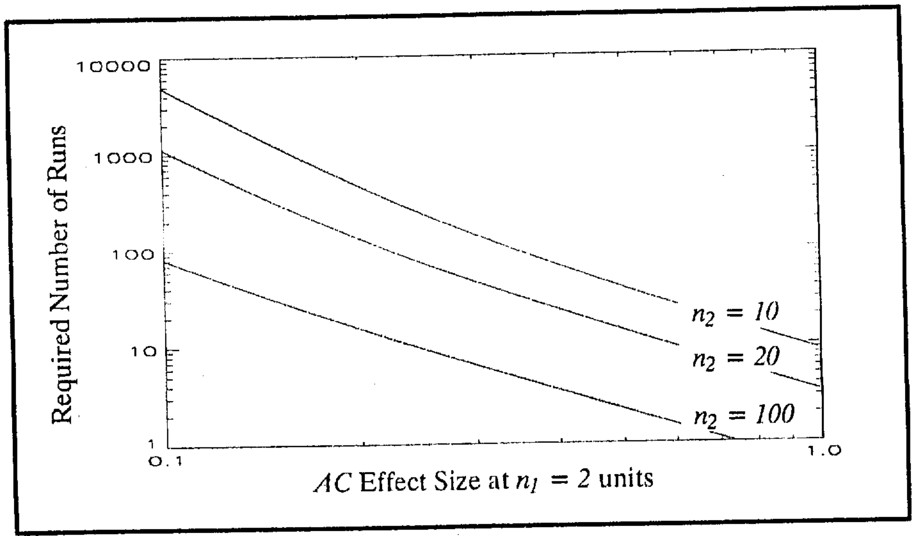

Figure 6. Runs Required for Biological *AP* Effect Sizes

We chose $n_1 = 2$ units for convenience. For example in a plant study, the physiological responses can easily be averaged over two plants and $n_2 = 10$ is within reason for a second data point. A unit could be a test tube containing cells or bacteria; the collection of all ten test tubes would simultaneously have to be the target of the *AP* effort to meet the constraints of a valid test.

The prospective tests we have described so far are conditional; that is, given an effect size, we provide a protocol to test if the mechanism for *AMP* is *PAP* or *DAT*. An unconditional test does not assume any effect size; all that is necessary is to collect data at a large number of different values of n, and fit a straight line through the resulting Z^2s. The mechanism is *PAP* if the slope is non-zero and may be *DAT* if the slope is zero.

Discussion

We now address the possible n-dependence of the model parameters. A degenerate case arises if ε_{AP} is proportional to $\sqrt{n}$; if that were the case, we could not distinguish between the *PAP* model and *DAT* by means of tests on the n dependence of results. If it turns out that in the analysis of the data from a variety of experiments, participants, and laboratories, the slope of a Z^2 vs n linear least-squares fit is zero, then either $\varepsilon_{AP} = 0.0$ or ε_{AP} is *exactly* proportional to $\sqrt{n}$ depending upon the precision of the fit (i.e., errors on the zero slope). An attempt might be made to rescue the *PAP* hypothesis by explaining the $\sqrt{n}$ dependence of Z^2 in the degenerate case as a fatigue or other time dependence effects. That is it might

be hypothesized that human participants might become *AP*-tired as a function of n; however, it seems improbable that a human-based phenomena would be so widely distributed and constant and give exactly the $\sqrt{n}$ dependency in differing protocols needed to imitate *DAT*. We prefer to resolve the degeneracy by wielding Occam's razor: if the only type of *AP* which fits the data is indistinguishable from *AC*, and given that we have ample demonstrations of *AC* by independent means in the laboratory, then we do not need to invent an additional phenomenon called *AP*. Except for this degeneracy, a zero slope for the fit allows us to reject all *PAP* models, regardless of their n-dependencies.

DAT is not limited to experiments that capture data from a dynamic system. *DAT* may also be the mechanism in protocols which utilize quasi-static target systems. In a quasi-static target system, a random process occurs only when a run is initiated; a mechanical dice thrower is an example. Yet, in a series of unattended runs of such a device there is always a statistical variation in the mean of the dependent variable that may be due to a variety of factors, such as Brownian motion, temperature, humidity, and possibly the quantum mechanical uncertainty principle (Walker, 1974). Thus, the results obtained will ultimately depend upon when the run is initiated. It is also possible that a second-order *DAT* mechanism arises because of protocol selection; how and who determines the order in tri-polar protocols. In second order *DAT* there may be individuals, other than the formal subject, whose decisions effect the experimental outcome and are modified by *AC*.

Finally, we would like to close with a clear statement of what is meant by *DAT*: the decisions on which experimental outcomes depend are augmented by *AC* to capitalize upon the unperturbed statistical fluctuations of the target system. In our follow-on paper, we will examine retrospective applications to a variety of data sets.

Acknowledgements

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Appendix

Mathematical Derivations for the Decision Augmentation Theory

In this appendix we develop the formalism for the Decision Augmentation Theory (*DAT*). We consider cases for the mean-chance-expectation (*MCE*), anomalous perturbation (*AP*), and anomalous cognition (*AC*) under two assumptions—normality and Bernoulli sampling. For each of these three models, we compute the expected values of Z and Z^2, and the variance of Z^{2*}

Mean Chance Expectation (*MCE*)

Normal Distribution

We begin by considering a random variable, X, whose probability density function is normal, (i.e., $N(\mu_0, \sigma_0{}^2)^\dagger$). After many unbiased measures from this distribution, it is possible to obtain reasonable approximations to μ_0 and $\sigma_0{}^2$ in the usual way. Suppose n unbiased measures are used to compute a new variable, Y, given by:

$$Y_k = \frac{1}{n}\sum_{j=1}^{n} X_{j,k}\,.$$

Then Y is distributed as $N(\mu_0, \sigma_n{}^2)$, where $\sigma_n{}^2 = \sigma_0{}^2/n$. If Z is defined as

$$Z = \frac{Y_k - \mu_0}{\sigma_n},$$

then Z is distributed as $N(0, 1)$ and $E(Z)$ is given by:

$$E^N_{MCE}(Z) = \frac{1}{\sqrt{2\pi}}\int_{-\infty}^{\infty} z e^{-0.5z^2} dz = 0. \qquad (1)$$

Since $Var(Z) = 1 = E(Z^2) - E^2(Z)$, then

$$E^N_{MCE}(Z^2) = \frac{1}{\sqrt{2\pi}}\int_{-\infty}^{\infty} z^2 e^{-0.5z^2} dz = 1. \qquad (2)$$

The $Var(Z^2) = E(Z^4) - E^2(Z^2) = E(Z^4) - 1$. But

* We wish to thank Zoltan Vassy for originally suggesting the Z^2 formalism.

† Throughout this appendix, this notation means:

$$N(\mu, \sigma^2) = \frac{1}{\sigma\sqrt{2\pi}} e^{-0.5\left[\frac{x-\mu}{\sigma}\right]^2}.$$

$$E^N_{MCE}(Z^4) = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{\infty} z^4 e^{-0.5z^2} dz = 3.$$

So

$$Var^N_{MCE}(Z^2) = 2. \qquad (3)$$

Bernoulli Sampling

Let the probability of observing a one under Bernoulli sampling be given by p_0. After n samples, the discrete Z-score is given by:

$$Z = \frac{k - np_0}{\sigma_0 \sqrt{n}},$$

where

$$\sigma_0 = \sqrt{p_0(1 - p_0)},$$

and k is the number of observed ones ($0 \le k \le n$). The expected value of Z is given by:

$$E^B_{MCE}(Z) = \frac{1}{\sigma_0 \sqrt{n}} \sum_{k=0}^{n} (k - np_0) B_k(n, p_0), \qquad (4)$$

where

$$B_k(n, p_0) = \binom{n}{k} p_0^k (1 - p_0)^{n-k}.$$

The first term in Equation 4 is the $E(k)$ which, for the binomial distribution, is np_0. Thus

$$E^B_{MCE}(Z) = \frac{1}{\sigma_0 \sqrt{n}} \sum_{k=0}^{n} (k - np_0) B_k(n, p_0) = 0. \qquad (5)$$

The expected value of Z^2 is given by:

$$E^B_{MCE}(Z^2) = Var(Z) + E^2(Z),$$

$$= \frac{Var(k - np_0)}{n\sigma_0^2} + 0,$$

$$E^B_{MCE}(Z^2) = \frac{n\sigma_0^2}{n\sigma_0^2} = 1. \qquad (6)$$

As in the normal case, the $Var(Z^2) = E(Z^4) - E^2(Z^2) = E(Z^4) - 1$. But*

* Johnson, N. L., and S. Kotz, *Discrete Distributions*, John Wiley & Sons, New York, p. 51, (1969).

$$E^B_{MCE}(Z^4) = \frac{1}{n^2\sigma_0^4}\sum_{k=0}^{n}(k - np_0)^4 B_k(n,p_0)$$

$$= 3 + \frac{1}{n\sigma_0^2}(1 - 6\sigma_0^2).$$

So,

$$Var^B_{MCE}(Z^2) = 2 + \frac{1}{n\sigma_0^2}(1 - 6\sigma_0^2) = 2 - \frac{2}{n}, \ (p_0 = 0.5). \qquad (7)$$

Anomalous Perturbation (*AP*)

Normal Distribution

Under the perturbation assumption described in the text, we let the mean of the perturbed distribution be given by $\mu_0 + \varepsilon_{ap}\sigma_0$, where ε_{ap} is an AP strength parameter, and in the general case may be a function of n and time. The parent distribution for the random variable, X, becomes $N(\mu_0 + \varepsilon_{ap}\sigma_0, \sigma_0{}^2)$. As in the MCE case, the average of n independent values of X, is $Y \sim N(\mu_0 + \varepsilon_{ap}\sigma_0, \sigma_n{}^2)$. Let

$$y = \mu_0 + \varepsilon_{ap}\sigma_0 + \Delta y,$$

where

$$\Delta y = y - (\mu_0 + \varepsilon_{ap}\sigma_0).$$

For a mean of n samples, the Z-score is given by

$$Z = \frac{y - \mu_0}{\sigma_n} = \frac{\varepsilon_{ap}\sigma_0 + \Delta y}{\sigma_n} = \varepsilon_{ap}\sqrt{n} + \zeta.$$

where ζ is distributed as $N(0, 1)$ and is given by $\Delta y / \sigma_n$. Then the expected value of Z is given by

$$E^N_{AP}(Z) = E_{AP}(\varepsilon_{ap}\sqrt{n} + \zeta) = \varepsilon_{ap}\sqrt{n} + E(\zeta) = \varepsilon_{ap}\sqrt{n}. \qquad (8)$$

and the expected value of Z^2 is given by

$$E^N_{AP}(Z^2) = E_{AP}([\varepsilon_{ap}\sqrt{n} + \zeta]^2) = n\varepsilon_{ap}^2 + E(\zeta^2) + 2\varepsilon_{ap}\sqrt{n}E(\zeta)$$
$$= 1 + \varepsilon_{ap}^2 n, \qquad (9)$$

since $E(\zeta) = 0$ and $E(\zeta^2) = 1$.

In general, Z^2 is distributed as a non-central X^2 with 1 degree of freedom and non-centrality parameter $n\varepsilon_{ap}{}^2$, $\mathrm{X}^2(1, n\varepsilon_{ap}{}^2)$. Thus, the variance of Z^2 is given by*

$$Var^N_{AP}(Z^2) = 2(1 + 2n\varepsilon_{ap}^2). \qquad (10)$$

Bernoulli Sampling

As before, let the probability of observing a one under MCE be given by p_0, and the discrete Z-score be given by:

* Johnson, N. L., and S. Kotz, *Continuous Univariate Distributions—2,* John Wiley & Sons, New York, p. 134, (1970).

$$Z = \frac{k - np_0}{\sigma_0 \sqrt{n}},$$

where k is the number of observed ones ($0 \leq k \leq n$). Under the perturbation assumption, we let the mean of the distribution of the single-bit probability be given by $p_1 = p_0 + \varepsilon_{ap}\sigma_0$, where ε_{ap} is an AP strength parameter. The expected value of Z is given by:

$$E^B_{AP}(Z) = \frac{1}{\sigma_0 \sqrt{n}} \sum_{k=0}^{n} (k - np_0) B_k(n,p_1),$$

where

$$B_k(n,p_1) = \binom{n}{k} p_1^k (1 - p_1)^{n-k}.$$

The expected value of Z becomes

$$E^B_{AP}(Z) = \frac{1}{\sigma_0 \sqrt{n}} \left[\sum_{k=0}^{n} kB_k(n,p_1) - np_0 \right]$$

$$= \frac{(p_1 - p_0)\sqrt{n}}{\sigma_0} = \varepsilon_{ap}\sqrt{n}. \qquad (11)$$

Since $\varepsilon_{ap} = E(Z)/\sqrt{n}$, so ε_{ap} is also the binomial effect size. The expected value of Z^2 is given by:

$$E^B_{AP}(Z^2) = Var(Z) + E^2(Z),$$

$$= \frac{Var(k - np_0)}{n\sigma_0^2} + \varepsilon_{ap}^2 n,$$

$$= \frac{p_1(1 - p_1)}{\sigma_0^2} + \varepsilon_{ap}^2 n.$$

Expanding in terms of $p_1 = p_0 + \varepsilon_{ap}\sigma_0$,

$$E^B_{AP}(Z^2) = 1 + \varepsilon_{ap}^2(n - 1) + \frac{\varepsilon_{ap}}{\sigma_0}(1 - 2p_0). \qquad (12)$$

If $p_0 = 0.5$ (i.e., a binary case) and $n \gg 1$, then Equation 12 reduces to the $E(Z^2)$ in the normal case, Equation 9.

We begin the calculation of $Var(Z^2)$ by using the equation for the jth moment of a binomial distribution

$$m_j = \frac{d^j}{dt^j}[(q + pe^t)^n] \Big|_{t=0}.$$

Since $Var(Z^2) = E(Z^4) - E^2(Z^2)$, we must evaluate $E(Z^4)$. Or,

$$E^B_{AP}(Z^4) = \frac{1}{n^2\sigma_0^4} \sum_{k=0}^{n} (k - np_0)^4 B_k(n,p_1).$$

Expanding $n^{-2}\sigma_0^{-4}(k - np_0)^4$, using the appropriate moments, and subtracting $E^2(Z^2)$, yields

$$Var(Z^2) = C_0 + C_1 n + C_{-1} n^{-1}. \qquad (13)$$

Where

$$C_0 = 2 - 36\varepsilon_{ap}^2 + 10\varepsilon_{ap}^4 + 8\frac{\varepsilon_{ap}}{\sigma_0}(1 - 2p_0)(1 - 2\varepsilon_{ap}^2) + 6\frac{\varepsilon_{ap}^2}{\sigma_0^2},$$

$$C_1 = 4\varepsilon_{ap}^2(1 - \varepsilon_{ap}^2) + 4\frac{\varepsilon_{ap}^3}{\sigma_0}(1 - 2p_0), \text{ and}$$

$$C_{-1} = 48 - 6[\varepsilon_{ap}^2 - 3]^2 + 12\frac{\varepsilon_{ap}^3}{\sigma_0}(1 - 2p_0) + \frac{(1 - 7\varepsilon_{ap}^2)}{\sigma_0^2} + \frac{\varepsilon_{ap}}{\sigma_0^3}(1 - 2p_0)(12p_0^2 - 12p_0 + 1).$$

Under the condition that $\varepsilon_{ap} \ll 1$ (a frequent occurrence in the perturbation approximation for AP), we ignore any terms of higher order than $\varepsilon_{ap}{}^2$. Then the variance reduces to

$$Var(Z^2) = 2 - 36\varepsilon_{ap}^2 + 8\frac{\varepsilon_{ap}}{\sigma_0}(1 - 2p_0) + 6\frac{\varepsilon_{ap}^2}{\sigma_0^2} + 4\varepsilon_{ap}^2 n + \frac{1}{n}\left[-6 + 36\varepsilon_{ap}^2 + \frac{(1 - 7\varepsilon_{ap}^2)}{\sigma_0^2} + \frac{\varepsilon_{ap}}{\sigma_0^3}(1 - 2p_0)(12p_0^2 - 12p_0 + 1)\right].$$

We notice that when $\varepsilon = 0$, the variance reduces to the MCE case for Bernoulli sampling. When $n \gg 1$, $\varepsilon \ll 1$, and $p_0 = 0.5$, the variance reduces to that derived under the normal distribution assumption. Or,

$$Var_{AP}^{B}(Z^2) \approx 2(1 + 2n\varepsilon_{ap}^2). \qquad (14)$$

Anomalous Cognition (*AC*)

The primary assumption for AC is that the parent distribution remains unchanged, (i.e., $N(\mu_0, \sigma_0{}^2)$. It further assumes that because of a AC-mediated bias the sampling distribution is distorted leading to a Z-distribution as $N(\mu_{ac}, \sigma_{ac}{}^2)$. In the most general case, μ_{ac} and σ_{ac} may be functions of n and time. Let ζ be given by

The expected value of Z is given by (by definition)

$$E_{AC}^{N}(Z) = \mu_{ac}. \qquad (15)$$

The expected value of Z^2 is given by definition as

$$E_{AC}^{N}(Z^2) = \mu_{ac}^2 + \sigma_{ac}^2. \qquad (16)$$

The $Var(Z^2)$ can be calculated by noticing that

$$\frac{Z^2}{\sigma_{ac}^2} \sim X_{nc}^2\left(1, \frac{\mu_{ac}^2}{\sigma_{ac}^2}\right).$$

So the $Var(Z^2)$ is given by

$$Var\left(\frac{Z^2}{\sigma_{ac}^2}\right) = 2\left(1 + 2\frac{\mu_{ac}^2}{\sigma_{ac}^2}\right)$$

$$Var_{AC}^{N}(Z^2) = 2(\sigma_{ac}^4 + 2\mu_{ac}^2\sigma_{ac}^2). \qquad (17)$$

As in the normal case, the primary assumption is that the parent distribution remains unchanged, and that because of a psi-mediated bias the sampling distribution is distorted leading to a discrete Z-distribution characterized by $\mu_{ac}(n)$ and $\sigma_{ac}{}^2(n)$. Thus, by definition, the expected values of Z and Z^2 are given by

$$E^{B}_{AC}(Z) = \mu_{ac}$$
$$E^{B}_{AC}(Z^2) = \mu^2_{ac} + \sigma^2_{ac}. \quad (18)$$

For any value of n, estimates of these parameters are calculated from N data points as

$$\hat{\mu}_{ac} = \frac{1}{N}\sum_{j=1}^{N} z_j \text{, and}$$

$$\hat{\sigma}^2_{ac} = \frac{N}{(N-1)}\left(\sum_{j=1}^{N}\frac{z_j^2}{N} - \hat{\mu}^2_{ac}\right).$$

The $Var(Z^2)$ for the discrete case is identical to the continuous case. Therefore

$$Var^{B}_{AC}(Z^2) = 2(\sigma^4_{ac} + 2\mu^2_{ac}\sigma^2_{ac}). \quad (19)$$